23 The Second Heart Sound

JOEL M. FELNER

Definition

The second heart sound (S_2) is a short burst of auditory vibrations of varying intensity, frequency, quality, and duration. It has two audible components, the aortic closure sound (A_2) and the pulmonic closure sound (P_2), which are normally split on inspiration and virtually single on expiration. S_2 is produced in part by hemodynamic events immediately following closure of the aortic and pulmonic valves. The vibrations of the second heart sound occur at the end of ventricular contraction and identify the onset of ventricular diastole and the end of mechanical systole.

Technique

The examination should be performed in a warm, quiet room in a manner identical to that described in Chapter 22, The First Heart Sound. Clinical assessment of S_2 is best performed with the patient lying comfortably in the supine position and breathing normally.

First, attempt to palpate the aortic and pulmonic components of the second heart sound in the second right and second or third left intercostal spaces (ICS), respectively. Then begin cardiac auscultation with the stethoscope placed at the second right ICS. The second sound, like the first, is evaluated by sequentially auscultating over the second left ICS, the fourth left ICS along the left sternal border, and the cardiac apex. When listening to the heart sounds, it is essential simultaneously to palpate either the carotid artery or apex impulse to determine the onset of systole.

The second heart sound is of shorter duration and higher frequency than the first heart sound. It has two audible components, the aortic closure sound (A_2) and the pulmonic closure sound (P_2), which must be separated by more than 20 msec (0.20 sec) in order to be differentiated and heard as two distinct sounds. It is clinically very important to determine the presence and degree of respiratory splitting and the relative intensities of A_2 and P_2.

Splitting is best identified in the second or third left ICS, since the softer P_2 normally is confined to that area, whereas the louder A_2 is heard over the entire precordium, including the apex. In order to appreciate splitting of S_2, it may be useful to gradually move the stethoscope ("inching") from the second right ICS to the fourth left ICS.

The influence of respiration on the second sound is extremely important. The examiner will wish to note respiratory variation both during quiet breathing and at times during exaggerated breathing. Slow, regular respirations are best for auscultation because a long deep breath may attentuate P_2 by interposing lung tissue over the stethoscope, and only a single sound will be heard. The interval between the two audible components of the second heart sound normally increases on inspiration and virtually disappears on expiration. The patient's age must be taken into consideration when assessing splitting of the second sound, since the likelihood of hearing a single S_2 during both respiratory phases increases with advancing age. As with the first sound, the patient should be examined in several positions. The supine position in young patients, for example, may yield an erroneous impression of abnormally wide S_2 splitting, which can be avoided by reexamining the patient in the sitting or standing position. The Valsalva maneuver may also be used to exaggerate splitting of the second sound.

When the second sound is split and both components can be heard and identified, a reliable judgment about the relative loudness (intensity) of each component can be made.

Basic Science

Rouanet, more than 140 years ago, attributed the second heart sound to closure of the aortic and pulmonic valves. Although this explanation has been generally accepted to the present time, several theories have been proposed to explain the genesis of the second heart sound. The most tenable to date suggests that closure of the aortic and pulmonic valves initiates the series of events that produces the second heart sound. The main audible components, however, result from vibrations of the cardiac structures after valve closure. Using high-fidelity, catheter-tipped micromanometers and echophonocardiography, it has been shown that the aortic and pulmonic valves close silently and that coaptation of the aortic valve cusps precedes the onset of the second sound by a few milliseconds. The second sound therefore originates from after-vibrations in the cusps and in the walls and blood columns of the great vessels and their respective ventricles. The energy from these oscillations comes from sudden deceleration of retrograde flow of the column of blood in the aorta and pulmonary artery when the elastic limits of the tensed valve leaflets are met. This abrupt deceleration sets the whole cardiohemic system into vibration.

In order to understand splitting of the second heart sound, knowledge of its relationship to the cardiac cycle is essential. A_2 and P_2 are coincident with the incisurae of the aorta and pulmonary artery pressure curves, respectively, and terminate left and right ventricular ejection periods. Right ventricular ejection begins prior to left ventricular ejection, has a slightly longer duration, and terminates after left ventricular ejection, resulting in P_2 normally occurring after A_2.

The differences between the aortic and pulmonary artery vascular impedance characteristics are also essential to understanding the effects of respiration on splitting of S_2. When the pressure curves of the pulmonary artery and right ventricle are recorded simultaneously, the pulmonary artery curve at the level of the incisura (dicrotic notch) lags behind the right ventricular curve, or "hangs out" after it. The duration of the "hangout interval" is a measure of

impedance in the pulmonary artery system. In the highly compliant (low-resistance, high-capacitance) pulmonary vascular bed, the hangout interval may vary from 30 to 120 msec, contributing significantly to the duration of right ventricular ejection. In the left side of the heart, because impedance is much greater, the hangout interval between the aorta and left ventricular pressure curves is negligible (less than or equal to 5 msec). The hangout interval therefore correlates closely with impedance of the vascular bed into which blood is being injected. Its duration appears to be inversely related to vascular impedance.

Alterations in the impedance characteristics of the pulmonary vascular bed and the right-sided hangout interval are responsible for many of the observed abnormalities of S_2. In a normal physiologic setting, inspiration lowers impedance in the pulmonary circuit, prolongs the hangout interval and delays pulmonic valve closure, resulting in audible splitting of A_2 and P_2. On expiration, the reverse occurs: pulmonic valve closure is earlier, and the A_2–P_2 interval is separated by less than 30 msec and may sound single to the ear. Since the pulmonary circulation has a much lower impedance than the systemic circulation, flow through the pulmonic valve takes longer than flow through the aortic valve. The inspiratory split widens mainly because of delay in the pulmonic component.

Traditionally it was believed that an inspiratory drop in intrathoracic pressure favored greater venous return to the right ventricle, pooling of blood in the lungs, and decreased return to the left ventricle. The increase in right ventricular volume prolonged right-sided ejection time and delayed P_2; the decrease in left ventricular volume reduced left-sided ejection time and caused A_2 to occur earlier. The delayed P_2 and early A_2 associated with inspiration, however, are best understood as an interplay between changes in the pulmonary vascular impedance and changes in systemic and pulmonary venous return. The net effect is that right ventricular ejection is prolonged, left ventricular ejection is shortened, and the A_2–P_2 interval widens during inspiration.

Clinical Significance

The clinical evaluation of the second heart sound has been called the "key to auscultation of the heart." It includes an assessment of splitting and a determination of the relative intensities of A_2 and P_2. Normally the aortic closure sound (A_2) occurs prior to the pulmonic closure sound (P_2), and the interval between the two (splitting) widens on inspiration and narrows on expiration. With quiet respiration, A_2 will normally precede P_2 by 0.02 to 0.08 second (mean, 0.03 to 0.04 sec) with inspiration. In younger subjects inspiratory splitting averages 0.04 to 0.05 second during quiet respiration. With expiration, A_2 and P_2 may be superimposed and are rarely split as much as 0.04 second. If the second sound is split by greater than 0.04 second on expiration, it is usually abnormal. Therefore, the presence of audible splitting during expiration (i.e., the ability to hear two distinct sounds during expiration) is of greater significance at the bedside in identifying underlying cardiac pathology than is the absolute inspiratory increase in the A_2–P_2 interval.

The respiratory variation of the second heart sound can be categorized as follows: (1) normal (physiologic) splitting; (2) persistent (audible expiratory) splitting, with normal respiratory variation; (3) persistent splitting without respiratory variation (fixed splitting); and (4) reversed (paradoxical) splitting.

Physiologic splitting is demonstrated during inspiration in normal individuals, since the splitting interval widens primarily due to the delayed P_2. During expiration, the A_2–P_2 interval is so narrow that only a single sound is usually heard.

Persistent (audible) expiratory splitting suggests an audible expiratory interval of at least 30 to 40 msec between the two sounds. Persistent splitting that is audible during both respiratory phases with appropriate inspirational and expirational directional changes (i.e., further increase of the A_2–P_2 interval with inspiration) may occur in the recumbent position in normal children, teenagers, and young adults. If these individuals sit, stand, or perform a Valsalva maneuver, however, the second sound often becomes single on expiration. In almost all patients with heart disease and audible expiratory splitting in the recumbent position, expiratory splitting persists when the patient is examined in the sitting or standing position. Thus, the finding of audible expiratory splitting in both the recumbent and upright positions is a very sensitive screening test for heart disease. Right bundle branch block (RBBB) is the most common cause of the persistence of audible expiratory splitting on standing.

Other causes of persistent expiratory splitting on standing may be due either to a delay in pulmonic valve closure or to early closure of the aortic valve. A delay in P_2 may be secondary to the following:

1. Delayed electrical activation of the right ventricle (e.g., left ventricular ectopic or paced beats, Wolff-Parkinson-White syndrome, and RBBB).
2. Decreased impedance of the pulmonary vascular bed (e.g., atrial septal defect, partial anomalous pulmonary venous return, and idiopathic dilatation of the pulmonary artery).
3. Right ventricular pressure overload lesions (e.g., pulmonary hypertension with right heart failure, moderate to severe valvular pulmonic stenosis, and acute massive pulmonary embolus).

An early A_2 may occur in patients with decreased resistance to left ventricular outflow (e.g., mitral regurgitation or constrictive pericarditis). Moderately large ventricular septal defects may also cause wide splitting of the second sound, but the aortic component is usually difficult to hear because of the loud holosystolic murmur.

Expiratory splitting of S_2 may occur in patients with severe congestive heart failure. The expiratory splitting usually disappears after satisfactory therapy of the heart failure. The high prevalence of expiratory splitting of S_2 in cardiomyopathy may be explained by a combination of low cardiac output, mitral regurgitation, pulmonary hypertension, right heart failure, and bundle branch block.

Fixed splitting denotes absence of significant variation of the splitting interval with respiration, such that the separation of A_2 and P_2 remains unchanged during inspiration and expiration. Atrial septal defect, with either normal or high pulmonary vascular resistance, is the classic example of fixed splitting of the second sound. The audible expiratory splitting in these patients is primarily a reflection of changes in the pulmonary vascular bed rather than selective volume overload of the right ventricle prolonging right ventricular systole. The fixed nature of the split is due to approximately equal inspiratory delay of the aortic and pul-

monic components, indicating that the two ventricles share a common venous reservoir. Respiratory splitting of the second sound immediately returns to normal following surgical repair of an atrial septal defect, although the pulmonic closure sound may remain delayed for weeks or months.

Severe right heart failure can lead to a relatively fixed split. This occurs because the right ventricle fails to respond to the increased volume produced by inspiration and because the lungs are so congested that impedance to forward flow from the right ventricle barely falls during inspiration. In anomalous pulmonary venous return without atrial septal defect, fixed splitting is not usually seen despite the simultaneous inspiratory delay in aortic and pulmonic closure.

The Valsalva maneuver may be used to exaggerate the effect of respiration and obtain clearer separation of the two components of the second sound. Patients with atrial septal defects show continuous splitting during the strain phase, and upon release the interval between the components increases by less than 0.02 second. In normal subjects, however, splitting is exaggerated during the release phase of the Valsalva maneuver. Variation of the cardiac cycle length may also be used to evaluate splitting of S_2. During the longer cardiac cycle, patients with atrial septal defect may show greater splitting as a result of increased atrial shunting and greater disparity between stroke volume of the two ventricles. In normal subjects, there is no tendency to widen the splitting with longer cardiac cycles.

Pulmonary artery hypertension causes variable effects on splitting of the second sound. Patients with ventricular septal defect who develop pulmonary hypertension may no longer have splitting of S_2. Patients with atrial septal defect and associated pulmonary hypertension maintain a wide and fixed split of S_2. Splitting is narrow (less than 30 msec), but remains physiologic, in patients with patent ductus arteriosus who develop pulmonary hypertension.

Paradoxical or reversed splitting is the result of a delay in the aortic closure sound. Therefore P_2 precedes A_2, and splitting is maximal on expiration and minimal or absent on inspiration. Identification of the reversed order of valve closure may be possible by judging the intensity and transmission of each component of the second sound. Often, however, the pulmonic component is as loud as the aortic component because of pulmonary artery hypertension secondary to left ventricular failure. The paradoxical narrowing or disappearance of the split on inspiration is a necessary criterion for diagnosing reversed splitting by auscultation.

Paradoxical splitting always indicates significant underlying cardiovascular disease and is usually due to prolongation of left ventricular activation or prolonged left ventricular emptying. The most common cause of paradoxical splitting of the second sound is left bundle branch block. Obstruction to left ventricular outflow of sufficient severity to delay aortic valve closure may also cause paradoxical splitting. In the context of aortic stenosis, such an auscultatory finding implies severe obstruction. Paradoxical splitting, however, occurs more commonly with hypertrophic cardiomyopathy than with aortic stenosis. Paradoxical splitting of the second sound may occur during the first few days after an acute myocardial infarction or secondary to severe left ventricular dysfunction.

A mistaken diagnosis of abnormal splitting of the second sound must be avoided. A late systolic click of mitral valve prolapse, the opening snap (OS) of mitral stenosis, a third heart sound (S_3), or a pericardial knock may be incorrectly thought to represent fixed splitting of the second sound. A systolic click may vary its location in systole with certain maneuvers that change the shape of the left ventricle (see Chapter 26, Systolic Murmurs). The best way to differentiate an A_2–P_2 from an A_2–OS is to have the patient stand up. The A_2–P_2 interval remains the same or narrows, whereas the A_2–OS interval widens. The third heart sound, which forms the S_2–S_3 complex, is lower in frequency than S_2, is best heard at the apex, is usually not heard at the basal auscultatory area, and occurs 0.12 to 0.16 second after A_2. The pericardial knock is a third heart sound that is slightly higher pitched and earlier than the usual S_3 and is also best heard at the apex.

The second sound can remain single throughout the respiratory cycle due to either absence of one component or to synchronous occurrence of the two components. Since the pulmonary vascular impedance increases with age, many normal patients over age 50 have a single S_2 or at most a narrow physiologic split on inspiration because P_2 occurs early. A single second sound, however, is usually due to inability to auscultate a relatively soft pulmonic component. Such inability is rare in healthy infants, children, and young adults and is uncommon even in older persons under good auscultatory conditions using a rigid stethoscopic diaphragm.

Hyperinflation of the lungs is perhaps the most common cause of inability to hear the pulmonic closure sound. All the conditions causing paradoxical splitting that delay A_2 may produce a single second sound when the splitting interval becomes less than 0.02 second. Inaudibility of P_2 due to a true decrease in its intensity is relatively rare, however, and suggests tetralogy of Fallot or pulmonary atresia. The pulmonic component may be inaudible in chronic right ventricular failure, or the aortic component may be masked by the systolic murmur in patients with aortic stenosis.

Pulmonary closure is completely fused with aortic closure throughout the respiratory cycle only in Eisenmenger's syndrome with a large ventricular septal defect or in cases of single ventricle, where the durations of right and left ventricular systole are virtually equal. The second sound may also be single in a variety of congenital heart defects (e.g., truncus arteriosus, tricuspid atresia, hypoplastic left heart syndrome, transposition of the great arteries, and, occasionally, corrected transposition of the great arteries).

The loudness of each component of the second heart sound is proportional to the respective pressures in the aorta and pulmonary artery at the onset of diastole. Dilatation of the aorta or pulmonary artery may also cause accentuation of the aortic and pulmonic components, respectively. The aortic component is normally of greater intensity than the pulmonic component. The aortic component, therefore, radiates widely over the chest, whereas the pulmonic component is heard mainly in the second left ICS with some radiation down the left sternal border. The greater radiation of the aortic component is probably due to the higher pressure in the aorta compared to that in the pulmonary artery. At any given level of pressure, however, the pulmonic component will be proportionately louder than the aortic component because of the closer proximity of the pulmonic valve and the pulmonary artery to the chest wall. These considerations account for the relative loudness of P_2 in young patients in whom the pulmonary arteries are quite close to the chest wall. They also account for the decreased intensity of both components of the second sound in patients with emphysema in whom both arteries are displaced from the chest wall.

The pulmonic component is considered to be abnormally

loud in a subject over age 20 if it is greater than the aortic component in the second left ICS or if it is audible at the cardiac apex. This may be due either to pulmonary artery hypertension or right ventricular dilatation, with part of the right ventricle assuming the position normally occupied by the left ventricle. A split second sound at the apex is, therefore, definitely abnormal. The loud P_2 commonly heard at the apex in patients with atrial septal defect is probably due to a dilated right ventricle encroaching upon the cardiac apex.

Decreased intensity of either component of the second sound may be due to a stiff semilunar valve, decreased pressure beyond the semilunar valve, or deformity of the chest wall or lung. A decreased intensity of P_2 is most common in patients with chronic obstructive lung disease or valvular pulmonic stenosis. A decreased intensity of A_2 is most common in patients with valvular aortic stenosis.

References

Adolph RJ, Fowler NO. The second heart sound: a screening test for heart disease. Mod Conc Cardiovasc Dis 1970;39:91–96.

Boyer SH, Chisholm AW. Physiologic splitting of the second heart sound. Circulation 1961;24:180–86.

Chandraratna PAN, Lopez JM, Cohen L. Echocardiographic observations on the mechanisms of the second heart sound. Circulation 1975;51:292–96.

Curtiss EL, Shaver JA, Reddy PS, et al. Newer concepts in physiologic splitting of the second heart sound. In: Leon DF, Shaver JA, eds. Physiologic principles of heart sounds and murmurs. New York: American Heart Association, 1975.

Harris A, Sutton G. The normal second heart sound. Br Heart J 1968;30:739–42.

Leatham A. Splitting of the first and second heart sounds. Lancet 1954;267:607–12.

Rouanet J. Analyse des bruits du coeur. Paris, Thesis No 252, 1832.

Shaver JA, Salemi R, Reddy PS. Normal and abnormal heart sounds in cardiac diagnosis. Part I: systolic sounds. In: O'Rourke RA, ed. Current problems in cardiology. Chicago: Year Book Medical Publishers, 1985;9–68.

Tavel ME. Clinical phonocardiography and external pulse recording, 2d ed. Chicago: Year Book Medical Publishers, 1972;38–41.

(See also references for Chapter 22, The First Heart Sound.)